Methods: Among 33 naïve chronic hepatitis B patients, the serum level Changes of sCD26 and sCD30 before, 1 and 3 months after the start of therapy with IFNα were evaluated using sandwich enzyme-linked immonosorbant assay. The success Rate of treatment with IFNα was also obtained in these patients.
Result: sCD26 serum level changes before the start of therapy till one month ($P = 0.001$) and three months ($P < 0.001$) after the start of therapy were related to success rate of therapy. sCD30 serum level changes were not related to treatment success.
Conclusion: Using changes of serum level of sCD26 might be useful in predicting the outcome of therapy in naïve chronic hepatitis B patients undergoing treatment with IFNα. More studies with longer follow up time in this topic are recommended.

OL-042 HBV particles preferably induce Kupffer cells to produce TGF-β1 over pro-inflammatory cytokines

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Background: Kupffer cells and related cytokines are believed to play a critical role in liver fibrosis. However, it is not clear what role Kupffer cells play in HBV-related fibrogenesis.
Methods: Primary rat Kupffer cells were cultured with different titers of HBV particles purified from the sera of chronic hepatitis B patients. The concentrations of TGF-β1, IL-6, IL-1 and TNF-α in the culture supernatant were measured every 24 hours for 7 days. The mRNA and protein levels of these cytokine in Kupffer cells were also analyzed by quantitative real-time PCR and Western blot, respectively.
Results: Kupffer cells maintained normal morphology and function throughout the 7-day HBV treatment. TGF-β1 secreted by Kupffer cells under stimulation with HBV at 6 Log IU mL^{-1} increased 5.38±4.54- and 7.75±4.27-fold by Days 3 and 7, respectively ($p < 0.01$). Western blotting showed the expression of TGF-β1 in Kupffer cells exposed to high titer HBV increased from 1.80±0.20- to 2.42±0.46-fold by Days 3 and 7, respectively ($p < 0.01$). In contrast, Kupffer cell expression and secretion of proinflammatory cytokines (IL-6, IL-1 and TNF-α) were unchanged throughout the experiment.
Conclusion: HBV could preferably stimulate Kupffer cells to produce profibrogenic/anti-inflammtory cytokine TGF-β1 over proinflammatory cytokines IL-6, IL-1 and TNF-α. This in *vitro* study may partly explain why overt liver fibrosis still presents in chronic HBV infection with minimal or even in the absence of necroinflammation.

OL-043 HBV DNA change between HBeAg positive and negative patients with chronic hepatitis B

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Background: Spontaneous decrease of HBV DNA among chronic hepatitis B (CHB) patients who were not treated with antivirus drugs had been reported. The aim of this study was to compare the HBV DNA change between HBeAg positive and negative CHB patients.
Methods: Dynamic change of HBV DNA level was observed for 12 weeks in 171 cases with admission HBV DNA level above 10,000 copies/mL. All patients had never been treated with antiviral or immunoregulatory drugs during this period. HBV DNA detection were carried out every two weeks. HBV DNA baselines, minimum level and changing degree of HBV DNA were compared respectively between the two groups. All the data were analyzed by software SPSS 13.0, *t*-test was used to compare means and difference was significant statistically when $P < 0.05$.
Results: Among HBeAg positive patients (n = 83, 48.5%), HBV DNA baseline, minimum level and changing degree of HBV DNA were (7.44±1.32) copies/ml, (3.59±0.99) copies/ml and (3.84±1.39) copies/ml, respectively; Among HBeAg negative patients (n = 88, 51.5%), HBV DNA baseline, minimum level and changing degree of HBV DNA were (7.26±0.96) copies/ml, (3.93±1.18) copies/ml, (3.35±1.070) copies/ml respectively. HBV DNA baselines had no significant difference statistically ($t = 0.759$, $P = 0.449$); minimum level of HBeAg positive patients was lower than that of HBeAg negative patients ($t = -2.020$, $P = 0.045$); changing degree of HBV DNA of HBeAg positive patients was greater than that of HBeAg negative patients ($t = 2.363$, $P = 0.027$).
Conclusion: Among patients with chronic hepatitis B underwent spontaneous HBV DNA decrease, HBeAg positive patients were more likely to have a greater HBV DNA change and lower minimum.

OL-044 'Dinucleotide-pattern' G→A hypermutations in the pre-core 5′-GGGG tetrad of HBe negative hepatitis B virus (HBV) variant

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Objective: Liver-APOBEC enzyme mediated G→A hypermutation has been reported in HBV genome that prefers 5′GGGG tetrad substrate as an antiviral-innate immune mechanism. We therefore, intended to analyze the occurrence of G→A hypermutations in the in the 5′GGGG tetrad of HBV pre-C coding sequences from HBeAg negative patients.
Methods: Six HBeAg seronegative hepatitis B patients with chronic liver disease were studied that fulfilled the inclusion criteria: presence of chronic hepatitis: persistence of HBsAg and anti-HBeAb seropositivity for at least 12 months, HBV DNA seropositivity, liver ALT level >1.5× upper limit of normal, and seronegativity for HCV and HDV. Viral DNA were extracted from the patient's sea and subjected to PCR-amplification, using pre-C/C specific primer sets followed by direct automated sequencing. The nucleotide sequences of HBe negative HBV variants were analyzed with that of wild type HBV, using the on line DNA multi-alignment program.
Results: The pre-C nucleotide sequence analysis of the six HBe negative viral variants showed classical G1896A mutations in 3 samples. Of these, one viral sequence showed an additional G1897A substitution, representing a 'dinucleotide-pattern' hypermutation resulting in pre-C stop codon (UGG→UAA) in the 5′GGGG tetrad. In another sample, a second G1899A substitution was also identified in the same tetrad stetch, but in the next codon (UGGGGC→UAGGAC).
Conclusion(s): (1) The pre-C 5′GGGG stretch appears as a hot-spot for G→A stop codon-mutations in the HBe negative chronic hepatitis B patients, and (2) This 'dinucleotide-pattern' G→A hypermutation are likely to be introduced by the host-APOBEC enzymes.